A TOXICITY INDEX FOR ASSESSING THE SUITABILITY OF STREAMS FOR AQUATIC LIFE

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by

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INTRODUCTION

Habitat quality, water quality, and biotic interactions all affect aquatic organisms. Streams have been classified according to the suitability of the water or habitat for aquatic life (water depth, flow velocity, substrate, temperature, concentration of oxygen and pollutants) or according to characteristics of the indigenous populations (species diversity, presence or absence of indicator species or guilds). Although some aquatic ecologists consider water quality a part of habitat quality, it is useful in this paper to make a distinction between chemical characteristics of the water which are measured routinely in water quality monitoring programs and the physical characteristics of the habitat which are not. While population data can indicate that a problem exists, the cause of the problem is not always easily determined from available information on water quality or habitat quality. If the causes are unknown, it is difficult to design measures to restore the biological quality of the stream. The biological significance of water quality data is often obscure, particularly because factors are usually considered one at a time (does the concentration of factor X exceed the water quality standard for aquatic life?), whereas organisms are exposed to many factors simultaneously. Water chemistry is usually measured in grab samples taken once a month, or even less frequently, so it usually is impossible to determine how long organisms were exposed to stressful factors. The degree of stress depends not only on concentration but also on duration of exposure.

The toxicity index described in this paper sums component toxicities

contributed by 20 common pollutants. The algorithms account for known effects of environmental factors which modify toxicity (temperature, dissolved oxygen, hardness or alkalinity). The toxicity index can be coupled to hydrologic and water quality models to estimate exposure durations as well as toxicity magnitudes, and to develop empirical relationships between index values, exposure durations, and fish community structure.

Although toxicity indices have been developed in both North America and Europe, research in Illinois has contributed substantially to verification, refinement and novel application of toxicity indices, including application to stream classification.

This paper describes: (1) the background, assumptions, and limitations of the toxicity index, (2) an example of the computation of the toxicity contributed by one component (ammonia), (3) application of the toxicity index to two rivers in Illinois, for the purposes of classifying reaches according to suitability for fish, determining which factors contribute the most toxicity, and for empirical determination of the relationship between the toxicity index and characteristics of the fish populations, and (4) future applications and improvements to the index.

ASSUMPTIONS AND LIMITATIONS

The Additive Assumption

Sprague and Ramsay (1965), Lloyd (1965), and Sprague (1970) were the first to propose the toxic units approach to predicting the joint toxicity of mixtures of common industrial and municipal pollutants. They expressed the separate toxicant concentrations as fractions of their lethal threshold concentrations and assumed the joint lethal effects were additive. The units were referenced to the species used to determine the lethal thresholds in

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laboratory bioassays. The bluegill is the reference organism in the Illinois toxicity index because it is widespread in Illinois (it is the state fish) and is commonly used in bioassays (Lubinski 1981; Lubinski and Sparks 1981; Lubinski et al. 1974; Muchmore et al. 1979; and Brigham and Hey 1981). If the concentration of zinc in water which kills 50% of the exposed population of bluegill sunfish (*Lepomis macrochirus*) in 96 hours is 8 mg/l (the 96-hour LC50), then 8 mg/l is considered to be 1.0 toxic unit, or more specifically, 1.0 bluegill toxicity unit,BGTU (Lubinski et al. 1974). A zinc concentration of 4 mg/l in a mixture therefore is 0.5 BGTU, and is the component toxicity attributable to zinc. If a stream contained 0.5 BGTU of zinc and 0.5 BGTU of copper, the water in this stream has a toxicity index value of 1.0 (0.5 + 0.5 = 1.0) and is predicted to be lethal to fish in 96 hours of exposure.

Two controversies developed over this approach. One was whether combinations of common pollutants were in fact additive, more than additive, or less than additive in contributing to lethality. The other was whether the toxicity index could be empirically related to sublethal effects on fish populations, e.g., failure of reproduction or changes in species occurrence or dominance.

The net results of many laboratory and field tests of the additive assumption are that very toxic mixtures (1.0 toxic unit) are more-than-additive, as measured by survival time; mixtures where component toxicities exceed 0.2 generally are additive, as measured by lethal thresholds; mixtures where component toxicities are less than 0.2 usually fail to add because the components apparently contribute no acute toxicity to the mixture; and reproducing populations of native fishes can be maintained where toxicity indices are less than 0.2, and habitat (cover, substrate, water depth and velocity) and biotic factors (food supply, predators, competitors, parasites

and disease organisms) are not limiting. These conclusions are supported by literature which will be reviewed briefly here, because of the central importance of the additive assumption to the toxicity index, and by the results of the two applications which are described later.

The archetypical example of a more-than-additive toxic effect was furnished by Doudoroff in 1952. He demonstrated that *Pimephales* sp. could withstand 8.0 ppm of zinc alone or 0.2 ppm of copper alone for 8 hours, but the combination of only 1.0 ppm zinc with 0.025 ppm copper killed most of the fish in 8 hours. This example has been cited frequently and used as a warning of the type of more-than-additive effects to be expected if more and more pollutants are introduced to the environment, without an assessment of their joint effects (Cairns 1957). More-than-additive effects of zinc-copper mixtures have also been demonstrated by other investigators (Lloyd 1961b).

It is therefore surprising that Herbert stated in 1965 that no cases had been found in which the toxicity of mixtures of poisons commonly found in sewage and industrial wastes (copper, zinc, lead, phenol, ammonia, and cyanide) were appreciably more than additive. This apparent contradiction is resolved by looking more closely at the concentrations and exposure times used in the experiments. As Sprague and Ramsay (1965) pointed out, survival times in mixtures where the total toxicity appreciably exceeds 1.0 toxic unit (Doudoroff's example) are shorter than expected. When lethal thresholds (96-hr LC50's or LT50's) are measured, the toxicities of mixtures of heavy metals and other toxicants generally do add up in laboratory experiments. However, there are exceptions as shown below.

Sprague (1970) pointed out that the toxicity of mixtures of phenol, ammonia, and zinc was overestimated by Brown et al. (1969) in three out of four cases where two of the toxicants were present at 0.10 to 0.14 toxic

units. When each toxicant was present at 0.2 toxic units or more, the toxicities did add up. These data suggest that the threshold for acute toxic effect postulated by Lloyd (1965) does exist, at least for metals, and that it may be approximately 0.2 toxic unit.

Results using mixtures other than metals alone are ambiguous. In Illinois Sparks and Anderson (1977) reported that a toxicity index underestimated the lethality of a mixture of linear alkyl sulfonate (LAS, a detergent), ammonia, and zinc by approximately 50%. In contrast, Esvelt et al. (1971) accurately estimated the toxicity of wastes entering San Francisco Bay by adding the toxic contributions of methylene blue-active substances (MBAS, mostly detergents) and ammonia. Herbert (1962) noted an 82.5 percent agreement between predicted and observed toxicities in field tests, although Sprague (1970) noted that the limit set for agreement was rather wide. In fresh-water reaches of four rivers in England, actual 48-hr LC50s approximated 65% of predicted values, and in the saline estuaries, prediction further underestimated actual toxicity (Brown et al. 1970). Lloyd and Jordan (1964) found their index consistently underestimated the toxicity of sewage effluents and that the relation between the predicted and observed toxicity was described by the function:

$$y = 1.25x - 0.59$$

where y is the observed toxicity and x the predicted toxicity.

Some of the underestimates of toxicity reported in the older literature probably result from bioassays which were run for arbitrary time periods, e.g., 8-, 24-, or 48-hours, which were too brief for full uptake of the toxicant and full exertion of the toxic effect in the test populations, particularly at the lower concentrations. The individuals in every test population differ in tolerance to the toxicant, and the difference is

expressed in survival time. The thresholds estimated from short-term bioassays could underestimate actual thresholds. Modern practice is to continue an acute bioassay for at least 96 hours, or better, until all mortality (or other toxic effect) has ceased for at least 24 hours.

Even with these limitations of the additive assumption however, most of the predicted toxicities of mixtures differed from the actual toxicities by no more than 50%. Considering that the modes of action of many of the toxicants are unknown, let alone the modes of interaction with other toxicants and with modifying factors in the water or in the organisms, it is remarkable that the error is not higher. It is likely that the error will be reduced as more is learned about interactions, which then can be incorporated in the toxicity index. Acceptance of a 50% error appears preferable to the alternative of not using the existing bioassay literature and chemical monitoring data to estimate the joint effects of toxicants in lakes and streams. In cases such as spills of complex wastes, where the predicted toxicity is several times greater than 1.0, a 50% error is insignificant. At the other extreme, field evidence from a variety of polluted rivers in England and Illinois supports a relatively narrow range of 0.2-0.4 for a threshold below which populations of native fishes can sustain themselves, if other factors are not limiting (this paper; Brigham and Hey 1981; Herbert et al. 1965; and Brown 1970).

Bioaccumulation

By definition, bluegill toxicity indices sum 96-hour LC50 values and thus pertain only to acute toxicity, or to empirically determined relationships between index values and fish populations in streams. However, similar indices can be based on chronic effects when sufficient data are developed. The index does not assess effects of toxicants which accumulate in organisms

from levels in water which are too low to have acute effects. The index therefore does not identify chemicals which might not affect fish but might affect consumers of fish.

Choice of Species and Life Stage

The present toxicity index is based on juvenile and adult life stages of the bluegill because most of the available toxicological information was for these stages. Additional toxicity indices, however, could be based on sensitive embryonic and larval stages of organisms (e.g. Reinbold and Pescitelli 1982) representing a variety of trophic levels or functional groups (e.g. Anderson et al. 1978) within aquatic systems. Multiple indices would be better predictors of species replacements or ecosystem-level effects.

Limitations of the Data Bases

Algorithms for computing component toxicities can be continually updated as more toxicity data become available on newly introduced chemicals and on interactions and modifying factors, but in the meantime, there are data gaps. For example, I could locate no information on the toxicity of fluoride to bluegills. Neuhold and Sigler (1960), however, reported 96-hr LC50s for fluoride-sensitive rainbow trout and fluoride-tolerant common carp, and it was reasonable to assume bluegill would be intermediate in sensitivity.

Automated samplers, event-triggered sampling (during a flood, drought, or spill), and water quality models (calibrated with available data) can overcome the limitations imposed by the relatively infrequent sampling (usually once a month, at best) characteristic of most water quality monitoring programs. It is particularly important to know the duration of exposure because fish can survive brief exposures to conditions (low oxygen, lethal concentration of a toxicant) which eventually would kill them. Another limitation of the water

quality data base is that total concentrations are measured, rather than toxic fractions. Doudoroff et al. (1966) demonstrated that molecular cyanide (HCN) is the toxic agent in solutions containing ionized cyanide (CN^-) and cyanide-metal complexes, but only total cyanide is measured by IEPA and it may not be reliable to compute the molecular fraction of complex cyanide solutions in streams, even when the general chemical composition and pH are known (Doudoroff 1976). If nontoxic cyanide complexes are present, use of the total cyanide concentration overestimates the toxicity. These limitations could be overcome with better analytical techniques or more sophisticated programs for calculating chemical equilibria in complex mixtures.

CALCULATION OF COMPONENT TOXICITIES: AN EXAMPLE USING AMMONIA

For some toxicants, the effects of environmental factors on the chemical form and concentration of the toxicant, and on the resistance of the aquatic organism, have been determined in the laboratory and can be accounted for in the toxicity index. An example, using ammonia, is described next.

Ammonia is a common pollutant in Illinois waters. It is formed by the breakdown of urea, and hence is found in effluents from livestock confinement areas and sewage treatment plants. Ammonia, in several chemical forms, is stored, transported by pipe, truck, rail and barge, and applied to agricultural lands as a nitrogen source for crops. It occurs in effluents from refineries and munitions industries.

Effects of Modifying Factors on Chemical Equilibria

Ammonia in water exists in a toxic, un-ionized form, NH_3, and a non-toxic ionized form, NH_4^+, with the equilibrium between the two forms governed by pH, temperature, and salinity, although in freshwater the salinity effect can be

ignored (Emerson et al. 1975). The total ammonia-nitrogen concentration (ionized + un-ionized) is measured in most water quality monitoring networks, but the proportion which exists in the toxic state can be determined from the field pH and temperature, using the tables or two equations provided by Emerson et al. (1975: 2382).

$$NH_3\text{-}N = \text{total ammonia-N} \times \frac{1}{1 + \text{antilog}(\text{pka} - \text{pH})}$$

where pka = the negative log of the ionization constant:

$$\text{pka} = -0.03229\ (\text{temp } {}^{o}C) + 10.05333$$

Effects of Modifying Factors on Sensitivity of the Fish

The toxicity of un-ionized ammonia to fish varies with the size of the fish and the temperature and dissolved oxygen concentration of the water (Roseboom and Richey 1977; Reinbold and Pescitelli 1981; Merkens and Downing 1957). The toxicity of ammonia increases at low temperatures probably because the overall metabolic rate of the fish is lower, and their ability to excrete ammonia in their urine is reduced. Fish pick up ammonia from the water via their gills, and form ammonia within their tissues as a waste product of protein metabolism. When the rate of ammonia uptake and production exceeds the rate of ammonia excretion, the internal concentration of ammonia rises to lethal levels (Fromm 1970; Brockway 1950). Small fish are more sensitive to ammonia than large fish, presumably because the ratio of gill surface available for ammonia uptake to body volume or mass is greater in the smaller fish. The toxicity of every chemical used in the toxicity index, including ammonia, increases at low dissolved oxygen concentrations, presumably because

low oxygen itself is a stressor, and virtually any reduction below saturation reduces the metabolic scope for excretion or detoxification. As ambient oxygen concentrations decline, some fishes can compensate to some extent by increased ventilation frequency or volume (Marvin and Heath 1968), but probably at the expense of other metabolic activities, including ammonia excretion.

The 96-hour LC50 for un-ionized ammonia (as NH_3-N) was regressed against fish weight (fwt) and water temperature (temp), using Illinois data of Reinbold and Pescitelli (1981) and Roseboom and Richey (1977). The lowest and highest temperatures used by these investigators were 4 and 28°C, and the regression equation is not extrapolated beyond the range of these data:

if temp < 4 °C, then the 96-hour LC50 = $10^{(.026639\ fwt + .025353(4) - .67645)}$

if temp > 28 °C, then the 96-hour LC50 = $10^{(.026639\ fwt + .025353(28) - .67645)}$

otherwise, the 96-hour LC50 = $10^{(.026639\ fwt + .025353(temp) - .67645)}$

Next, the LC50 is adjusted to reflect the increased toxicity of un-ionized ammonia at dissolved oxygen (DO) concentrations below saturation (Merkens and Downing 1957):

LC50 = LC50 (at 100% saturation) x .013297 (DO, % saturation) - .32965

Unfortunately, only 2 levels of dissolved oxygen were tested. I assumed a linear relationship between the LC50 and DO.

Application to an Ammonia Spill in the Illinois River

For the purposes of this example, fish weight is set at 1 gram, which would probably be at the lower end of the average weight for bluegills in their first year of life in January (Carlander 1977) when 622,000 gallons of urea ammonium nitrate solution spilled into the Illinois River at Seneca, Illinois. A dispersion model indicated that the total ammonia concentration

6.5 river miles downstream, at Marseilles, was 47 mg/l for approximately 19 hours (personal communication, 8 April 1988, Mr. Thomas Butts, Professional Scientist and Assistant Head, Water Quality Section, Illinois State Water Survey, Peoria, Illinois).

Sensitivity of Ammonia Component Toxicity to Modifying Factors. Dissolved oxygen levels have a marked effect on the predicted toxicity of 47 mg/l total ammonia-N (Table 1). At saturation, the ammonia spill exceeded twice the lethal concentration, but at 13% saturation, the component toxicity was greater by 3 orders of magnitude, and would probably have killed fish within a few hours at summer temperatures. At colder temperatures, less of the total ammonia exists in the toxic, un-ionized form, but the sensitivity of the fish increases, so the net change in component toxicity is rather small, from 2.69 to 1.90, as the temperature declines from 28 ^{o} C to 4 ^{o} C (Table 1). At cold temperatures, fish may be exposed to ammonia longer than it takes a spill to pass a fixed point, because they lose their equilibrium and float upside down (Reinbold and Pescitelli 1981) and thus would be carried along in the spill.

Conclusions

Even assuming the DO was at saturation, and no other toxicants were present, the toxicity attributable to the spill was approximately twice the lethal threshold at the cold temperatures expected in January. Fish which lost their equilibrium were exposed to these lethal concentrations longer than 19 hours, and very probably died.

The component toxicity approach was a rational way of integrating information on environmental factors, chemical concentrations, and susceptibility of organisms to assess the probable degree of harm to aquatic life caused by this ammonia spill. The next application examines toxicities

contributed by 20 components in the Illinois River and the connecting Chicago-area canals, collectively known as the Illinois Waterway.

APPLICATION TO THE ILLINOIS WATERWAY

The Illinois Waterway extends from the highly altered rivers and waterways in Chicago downstream 326 miles via the Illinois River to the Mississippi River upstream of St. Louis. The numbers and kinds of native gamefish and their condition declines in the upstream direction, towards Chicago, where fish populations are dominated by a few species, including the introduced carp and goldfish (Sparks and Starrett 1975).

Procedures

In the years 1972-1974, the toxicity index was applied to the Illinois Waterway, to determine whether high toxicity values were associated with the degraded fish populations in the upstream reaches, and to determine what components contributed the most toxicity. Field tests, using bluegills exposed to river water for 4 days in cages in the river and in aerated plastic pools on shore (where the water was renewed by pumping), were conducted at an upstream (Dresden) and a downstream (Beardstown) site in 1974 (Lubinski et al. 1974). Controls were maintained nearby in cages in less polluted tributaries.

Water quality data were obtained from the Illinois Environmental Protection Agency (IEPA) for samples taken 4 to 13 times per year at 20 stations along the Illinois Waterway. The same data were obtained by the Natural History Survey on samples taken at least once daily during the field tests.

Results

Field bioassays. The field bioassays confirmed that no short-term mortalities attributable to toxicity occurred in the two reaches of the Illinois River or in the tributaries where the daily index values were less than 0.14 (Lubinski et al. 1974). No mortalities occurred at Dresden. At the Beardstown site, at the mouth of the Sangamon River, there were no mortalities in the aerated swimming pool receiving Illinois River water, but 22% of the 50 test fish confined in the cage in the Illinois River died, probably because of the stress of swimming against a relatively high current velocity (0.4-0.6 m/sec) or being forced against the mesh by the current (Lubinski et al. 1974). Twelve percent of the fish died in a backwater area receiving flow from the Sangamon River where the water levels were falling and the cage had to be lifted from the mud and moved to deeper water several times, stressing the fish.

Mean Toxicity Indices. The mean annual toxicity indices in the Illinois Waterway (the sum of the toxicity indices at each station, divided by the number of samples taken that year) generally were below 0.1 at the station farthest upstream, which receives clean water from Lake Michigan via a navigation lock, well above 0.2 through the Chicago waterways, and 0.1 or less starting where the Chicago Sanitary and Ship Canal joins the Des Plaines River (Figure 1). The year 1972 was an exception, with elevated toxicity indices at Peoria.

Maximum Toxicity Indices. Extreme events inevitably go undetected because of the small number of water quality samples taken per year by the U. S. Geological Survey and the Illinois Environmental Protection Agency (12 or less). If it takes approximately 5 minutes to fill the sample bottle each month, there are 43,195 minutes in which no samples were taken (60 minutes x

24 hours x 30 days = 43,200, less 5 minutes = 43,195). The sample represents conditions occurring during only 1/10,000th of the month. Maximum annual toxicity indices partially compensate for this limitation by adding the peak toxicities contributed by each component during the year. The assumption is that the highest component toxicities occurred simultaneously, and the maximum index approximates "worst case" conditions. Such an assumption is not completely unreasonable because: (1) if one toxicant is at high concentration because of an excessive discharge, several others usually are too, because industrial wastes typically are complex mixtures of pollutants, and (2) during low flow conditions, all wastes are more concentrated because there is less dilution capacity. (A better, but more costly simulation procedure, in terms of data and programming requirements, which does not make the "simultaneous maxima" assumption, is described in application 2 below.) Maximum toxicity indices approach 0.5 in the upper Illinois Waterway (except for the uppermost station, near Lake Michigan), and generally decline downriver, with the exception of the Peoria stations in 1972 (Figure 1).

Component Toxicity: Cyanide. The 1972 peak in toxicity at Peoria is explained largely by the cyanide component (Figure 2), with some contribution from zinc and copper (relatively high values, compared to other years, did occur together in one sample in this case). The spiky, highly variable cyanide pattern is typical of toxicants which are accidentally or sporadically introduced, and it is fortuitous that the monthly sampling in 1972 happened to detect an apparent spill which was moving downstream, and which probably originated from an industrial source in Peoria.

Component Toxicity: Ammonia. The dominant contributor to the pattern of high toxicity in the Chicago waterways and declining toxicity downstream is ammonia (Figure 2), which is continuously released from the sewage treatment

plants in Chicago. Ammonia probably declines downstream because of dilution by tributaries, uptake by aquatic plants in the upper Illinois Waterway, and conversion to nitrate by bacteria in the water. Toxicity in portions of the Illinois Waterway in Chicago could be reduced below an index of 0.2 by increased diversion of clean dilution water from Lake Michigan, but the diversion increase would have some potentially deleterious effects, including possible scouring of toxicant-laden sediments from the waterways downstream into more biologically productive areas (Havera et al. 1980).

Despite improvements in sewage treatment in the Chicago River since the early 1970s (Macaitis et al. 1987), ammonia remains a problem in much of the Illinois River. Unpublished data from the U. S. Fish and Wildlife Service (personal communication, 29 February 1988, Richard Ruelle, Aquatic Toxicologist, Environmental Services Section, U. S. Fish and Wildlife Service Field Office, Rock Island, Illinois) indicates that sediments in backwaters at least as far as 200 miles downstream from Chicago contain ammonia which is released in sufficient quantities when the sediments are agitated to kill fish in 96-hour laboratory bioassays. Sediments are frequently agitated by wind- and boat-generated waves (Jackson and Starrett 1959; Sparks and Starrett 1975). Sparks (1984) reported that un-ionized ammonia concentrations in excess of the lethal level for fingernail clams (0.06 mg/l NH_3-N) occurred in the Illinois River at Marseilles, Hennepin and Lacon in 1980, probably because uptake of CO_2 and HCO_3- during algal blooms raised the pH above 8 and increased the proportion of ammonia existing in the toxic un-ionized form. Fingernail clams are an important food for diving ducks and bottom-feeding fish, both of which were adversely affected by the die-off of the clams and other mud-burrowing invertebrates in a 100-mile reach of the Illinois River in 1958 and by their continuing failure to recolonize (Mills et al. 1966; Sparks

1984).

Summary of Application 1

In summary, the toxicity index proved useful in interpreting the water quality data collected by the IEPA on the Illinois Waterway to determine what toxicants are acutely limiting to fish and should be controlled to restore fish populations. Index values above 0.2 in the upper Illinois Waterway are associated with depauperate fish populations in generally poor body condition. The toxicity index falls below 0.2 downstream, where more species of native fishes, in better condition, occur. The principal contributor to the total acute toxicity is ammonia, with sporadic contributions from cyanide. Ammonia originates from such a widely dispersed, large capacity sewage system (Chicago) that pulses are absorbed and ammonia loading is relatively constant, but downstream variations in temperature, pH, and dissolved oxygen alter the component and overall toxicities. Therefore, an important limitation was the infrequency of water quality sampling (only 4 to 13 times per year), which made it difficult to detect extreme conditions or determine how long fish were exposed. The next application describes how water quality modeling can be used to overcome this limitation and, when coupled with the toxicity index, used to predict the effects of alternative pollution control measures on fish populations.

APPLICATION 2: EVALUATING STRATEGIES FOR WATER QUALITY MANAGEMENT

Objectives

The objectives of this project were (1) to explore the relationship between fish communities and their physical and chemical environments, using the good water quality and fish population data sets and a calibrated water quality model available for the DuPage River in northeastern Illinois, (2) to

develop a continuous toxicity function for relating the status of these communities to spatial and temporal patterns of physical and chemical events, and (3) to use the resulting toxicity functions to predict effects of alternative pollution control measures on fish populations (Brigham and Hey 1981).

Procedure

Three stream reaches of the DuPage River were selected for modeling and analysis, based on known differences in fish faunas. One reach supported a mixed community of 17 species, including bluegill and carp, (hereafter referred to as the bluegill reach), one supported only 6 species, excluding bluegill and dominated by carp (carp reach), and the third reach was fishless (Brigham and Hey 1981). The toxicity index was calculated for each reach at 1-hour intervals over a simulated span of 3 years, using water quality values generated by hydrologic and water quality models which were calibrated for the reaches. The models were developed by Hydrocomp, Inc., Palo Alto, California and implemented by the Northeastern Illinois Planning Commission. The toxicants were un-ionized ammonia, cyanide, lead, zinc, copper, linear alkyl sulfonate detergents (LAS), and total residual chlorine (TRC). After initially observing the magnitude and variability of the toxicity function generated for each reach (Figure 3), certain water quality input variables were modified to simulate different management practices on each stream reach.

Results

Critical Thresholds. The toxicity indices for the three stream reaches on 17 March 1972 are typical of the run simulating the period from 1 October 1970 to 30 September 1973 (Figure 3). Toxicity levels regularly exhibited in

these reaches compared well with the results of previous investigators, who indicated that the effects of acute toxicity in altering the species composition of fish communities became measurable in streams at levels of 0.2 to 0.4 toxic units (Lloyd and Jordan 1964; Edwards and Brown 1966).

Relationships between Frequency, Duration and Intensity of Exposure and Fish Populations. Perhaps more important than the typical values however, are the frequency and duration of episodes where toxicities exceeded the lethal value of 1.0 and the empirically determined threshold of 0.2-0.4 (Table 2). In the fishless reach, the toxicity index made 194 excursions above 3.0 lasting 1 hour or more during the 3 years, 39 excursions lasting 24 hours or more, and 21 lasting 96 hours or more. The carp reach exhibited toxicity levels of 1.0 unit for 96 hours or more on 14 occasions, whereas the bluegill reach never exceeded 0.3 for even 1 hour. There were only 2 occasions during the simulated 3-year period when the toxicity index in the bluegill reach was between 0.25 and 0.30 for 24 hours or more. The average length of these 2 excursions was 36.5 hours (Brigham and Hey 1981).

Simulation of Management Alternatives

Assume a management goal of changing the fishless reach to a reach capable of supporting fish. A typical management plan might include: (1) reduction of ammonia concentrations from wastewater treatment plants to 1.5 mg/l during the summer and 4.0 mg/l during winter, (2) elimination of combined (stormwater and sewage) sewer overflows, (3) reduction of sediment oxygen demand, and (4) moderate increase of dissolved oxygen in the wastewater effluents or in the stream (Brigham and Hey 1981).

This plan primarily targets ammonia toxicity, which is reduced by 3 orders of magnitude, from 20.2 to 0.023 (Table 3). The mean toxicity index declines by an order of magnitude, from 23.0 to 2.12 (Table 3), but still

significantly exceeds the mean index of 1.04 in the carp reach (Brigham and Hey 1981). The largest remaining contributor to the total toxicity is chlorine (mean component toxicity = 1.94). Cessation of effluent chlorination would reduce the mean index to 0.186, which is very close to the mean of 0.115 in the bluegill reach. If no excursions above 0.3 occurred, and no factors other than toxicity are limiting, a mixed community of native fishes probably could be maintained.

This simulation was run in 1981, and the Illinois Environmental Protection Agency has since abandoned the requirement for effluent chlorination, based on evidence that there would be little additional public health risk from infectious diseases and much improvement in water quality for aquatic life. The latter evidence included studies which employed or referred to the toxicity index (Muchmore et al. 1979; Dreher 1981; Hey, Pappas and Cox 1980; and Hey et al. 1982). Fish populations in the Chicago waterways have shown recent improvement following discontinuation of effluent chlorination (personal communication, 1 March 1988, Mr. Samuel Dennison, Fisheries Biologist, Metropolitan Sanitary District of Greater Chicago).

FUTURE DEVELOPMENT

The toxicological data base for the index was last revised in March 1981. The programs for computing toxicity indices from IEPA water quality data are written in BASIC for a Tektronix 4051 microcomputer and a CYBER 175 at the University of Illinois (Lubinski 1981). The University is replacing the CYBER and the Tektronix 4051s are no longer in common use. The algorithms should be rewritten, using updated data, for IBM- or Apple-compatible personal computers, and a new user's guide prepared.

The revised index then should be applied to reaches intermediate in toxicity between the bluegill reach and the carp reach of the DuPage River study (Brigham and Hey 1981) to quantify more precisely the timing, frequency, duration, and intensity of exposures which cause shifts in community structure. The differences in toxicity and exposure patterns between the bluegill and carp reaches on the DuPage River were too great to determine, for example, whether the community structure characteristic of the bluegill reach would degrade if the index exceeded 0.3 for brief periods.

This type of information is useful in pollution control engineering, where a performance standard is achieved within some specified variation and failure rate. If the biological consequences of excursions beyond the mean can be specified, then waste control programs can be designed to stay within the limits without incurring unnecessary expense to achieve lower variation or failure rates.

The timing of excursions also should be examined, e.g. do excursions in the spring when larval fish are present have greater effect on fish populations than the same excursions in late summer? If so, the waste loading could be adjusted seasonally to protect aquatic life in the stream. Although the present index, which is based on toxicity to adult fish, can be related empirically to the status of fish populations in streams, as described in the above applications, another approach would be to develop toxicity indices for sensitive life history stages and use them at the appropriate season.

The present index is based on concentrations of toxicants in water. In the Illinois River, water quality has improved without a concomitant recovery of infaunal macroinvertebrate communities, because of an apparent legacy of toxicants remaining in the sediments (Sparks 1984). Toxicity indices should be developed for reference species representing several trophic levels and

functional groups, used together to increase the reliability of simulations and estimations, and verified by field trials or by application to data-rich environments, as described in this paper.

In particular, indices should be developed for assessing the quality of sediments, as well as quality of water, using benthic macroinvertebrates and rooted aquatic macrophytes as reference organisms. Sediment LC50s (in mg of toxicant per kg of "standard" sediment materials, e.g. montmorillonite clay or natural sediments of consistent composition) could be determined by adding reagent grade toxicants to a sediment slurry, allowing it to settle, then adding the test organisms. The additive assumption should be tested with mixtures of toxicants in sediments. The database on sediment LC50s would be employed in a sediment toxicity index just as described above for the water-based index.

SUMMARY

The toxicity index provides a way of relating water quality monitoring data to toxicity data available in the literature and to fish populations in streams, so that stream reaches can be classified according to their suitability for fish communities of varying sensitivity to common pollutants and environmental stressors. The index goes beyond classification however, to identification of causative factors. Chemical concentration units are converted to toxicity units, so the chemicals which contribute the most toxicity in a stream reach can be identified. The component toxicities also can be summed to provide an estimate of the total toxicity in a reach. The assumption of additive effects appears generally valid if lethal thresholds, rather than survival times, are used to measure toxicity. Predicted lethal thresholds are generally within 50% of measured thresholds in laboratory and

field experiments where complex mixtures are present.

Where sufficient data exist, the effects of factors which modify chemical equilibria or the sensitivity of fish (temperature, pH, dissolved oxygen, calcium concentration or hardness) can be taken into account in the algorithms. One indirect, beneficial result of this systematic search for information on interactions is that toxicological data gaps and research needs are identified and prioritized.

Application of the toxicity index to 1970s water quality data from the Illinois River indicates that ammonia from the Chicago area is a major contributor to toxicity in the upper river, with sporadic contributions from cyanide downstream of Peoria. Mean index values above 0.2 occur in upstream reaches where there are depauperate fish communities dominated by introduced carp and goldfish. Native fish increase downstream where mean and "worst case" index values are generally below 0.2. The index also was used in an after-the-fact analysis to determine that a 1988 spill of urea ammonium nitrate into the upper Illinois River probably killed fish several miles downstream.

Toxicity indices in the range 0.2-0.4 have been established as the threshold for alteration of the species composition of fish communities by results of field tests in the Illinois River and in several English rivers and by analysis of toxicity simulations in 3 reaches of the Dupage River: a fishless reach, a carp-dominated reach, and a reach with a mixed community including the native bluegill sunfish. A time dimension was added to this threshold in the DuPage River study, where the index never exceeded a sublethal value of 0.30 for even 1 hour in the bluegill reach, there were only 2 excursions between 0.25 and 0.30 (lasting an average of 36.5 hours), and the average toxicity was 0.115 during a simulated 3-year period.

Toxicity indices are not designed to assess bioconcentration effects or to be used in place of direct in-plant or in-stream toxicity testing programs. The projects described here, however, have demonstrated that these indices can be used to complement water quality monitoring programs by providing numerical values that describe a biological parameter (toxicity). The recent regulatory emphasis that has been placed on effluent toxicity testing and biological monitoring suggests that the results of water quality monitoring programs are of limited value in assessing toxicity problems. Although it is true that a limited number of in-stream measurements for a particular toxicant should not be used in sole support of any important water management decision, the alternative of not considering these data at all seems equally unacceptable and, in fact, undermines a common objective of water quality monitoring programs, which is to provide information on which to base management decisions.

Unfortunately, the products of most water quality monitoring programs are voluminous tables of data, which decision makers find difficult to interpret. Analysis usually is confined to the number of times standards for individual constituents were exceeded rather than to interactions and their biological consequences. Toxicity indices provide a logical way to assess the joint action of toxicants and the modifying effects of environmental factors on aquatic organisms.

As demonstrated by the projects described here, toxicity indices can be used to determine which chemical components contribute the most toxicity at a given location or time, to relate temporal and spatial variations in water quality to fish community structure, to evaluate alternative pollution control strategies, to assess the biological effects of spills, and to classify stream reaches according to their suitability for fish.

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Dr. Kenneth S. Lubinski, currently with the U. S. Fish and Wildlife Service in LaCrosse, Wisconsin, contributed substantially to various projects involving toxicity indices from 1973 to 1974, and again from 1979 to 1987.

K. Douglas Blodgett, Illinois Natural History Survey, River Research Laboratory, Havana, wrote a program in LOTUS to calculate ammonia component toxicity, using updated information, for analysis of the 3 January 1988 ammonia spill in the Illinois River.

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FIGURE LEGENDS AND LIST OF TABLES FOR TOXICITY INDEX MANUSCRIPT BY S

Figure 1. Mean and maximum toxicity indices in the Illinois Waterway, 1972-1974. River mileages begin downstream at the confluence with the Mississippi, at river mile 0, and progress upstream toward Chicago and Lake Michigan, at river mile 330. The horizontal line is drawn at a toxicity index value of 0.2, below which populations of native species can maintain themselves, if factors other than acute toxicity are not limiting. An index of 1.0 is lethal, equivalent to the 96-hour LC50. See text for explanation.

Figure 2. Cyanide and ammonia component toxicities in the Illinois Waterway, 1972-1974. River mileages begin downstream at the confluence with the Mississippi, at river mile 0, and progress upstream toward Chicago and Lake Michigan, at river mile 330. The horizontal line is drawn at a toxicity index value of 0.2, below which populations of native species can maintain themselves, if factors other than acute toxicity are not limiting. An index of 1.0 is lethal, equivalent to the 96-hour LC50. See text for explanation.

Figure 3. Toxicity indices at 1-hour intervals in 3 reaches of the DuPage River on 17 March 1972. Indices are output from a simulation model, calibrated for the DuPage River.

Table I. Toxicity of a total ammonia-N concentration of 47 mg/l in the Illinois River under different conditions.

Table 2. The number of excursions past different toxicity levels in the DuPage River during a simulated 3-year period.

disk: COMPAQ HARDCARD, PRO 2, and BLANK TRANSFER file: BLUEGILL.MNS

Table 3. Impact of a water quality management plan on toxicity in the DuPage River.

TABLE 1 -- Toxicity of a total ammonia-N concentration of 47 mg/l to 1-gram bluegills under different conditions of temperature (^{o}C), pH, and dissolved oxygen (DO).

Temp	pH	DO		Bluegill Toxicity Index
		mg/l	% Saturation	
28	8.0	7.5	100	2.69
28	8.0	5.0	66	4.83
28	8.0	3.0	40	13.4
28	8.0	1.0	13	3080
28	7.0	5.0	66	0.514
28	7.5	5.0	66	1.60
28	8.0	5.0	66	4.83
28	8.5	5.0	66	13.4
4	8.0	12.2	100	1.90
8	8.0	11.1	100	2.05
12	8.0	10.1	100	2.22
4	8.0	8.1	66	3.43
4	8.0	4.9	40	9.33
4	8.0	1.6	13	534

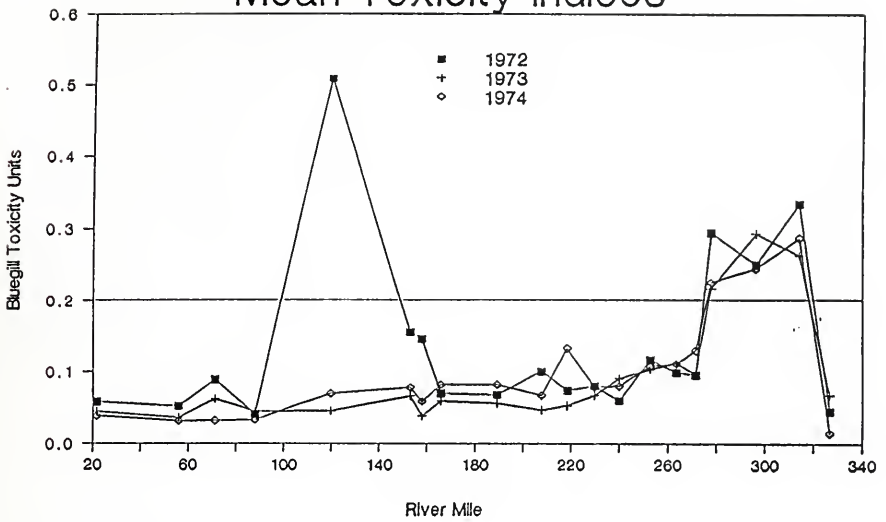
TABLE 2 -- The number of excursions past different toxicity levels for various lengths of time during a 3-year period, 1 October 1970 to 30 September 1973, in 3 reaches of the DuPage River (Lubinski 1981).

	Toxicity Index BGTUs	Number of Toxicity Index Excursions for 3 Duration Times		
		1 h	24 h	96 h
Fishless reach	1.0	73	51	42
	2.0	155	55	34
	3.0	194	39	21
Carp reach	1.0	74	31	14
	2.0	16	1	0
	3.0	12	1	0
Bluegill reach	0.1	194	29	15
	0.2	37	25	11
	0.3	0	0	0

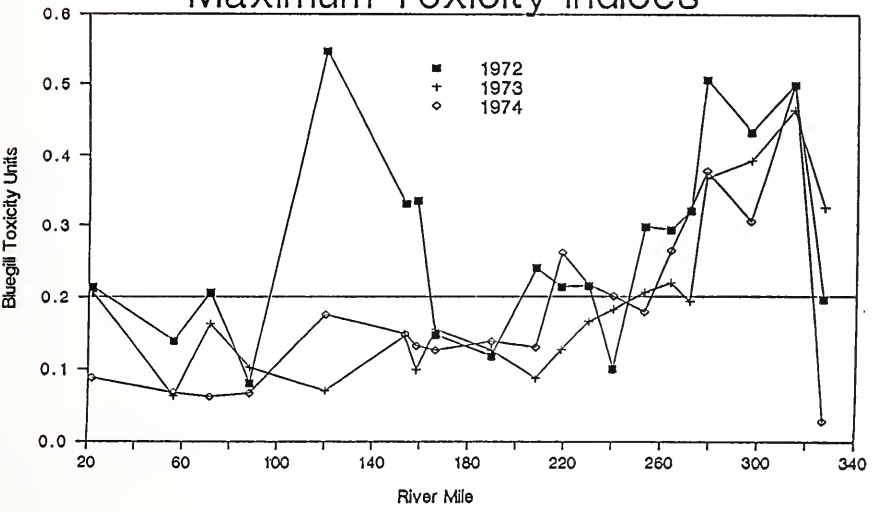
TABLE 3 -- Impact of a hypothetical water quality management plan (see text for explanation) upon the toxicity index in the fishless reach, DuPage River, DuPage County, Illinois (Brigham and Hey 1981).

	Without Plan	With Plan
Toxicity Index		
max.	784	51.5
min.	0.120	0.118
mean.	23.0	2.12
Component Toxicity		
Ammonia	20.2	0.023
Cyanide	0.088	0.079
Lead	0.023	0.000
Zinc	0.004	0.003
Copper	0.020	0.008
LAS	0.772	0.073
Chlorine	1.94	1.94

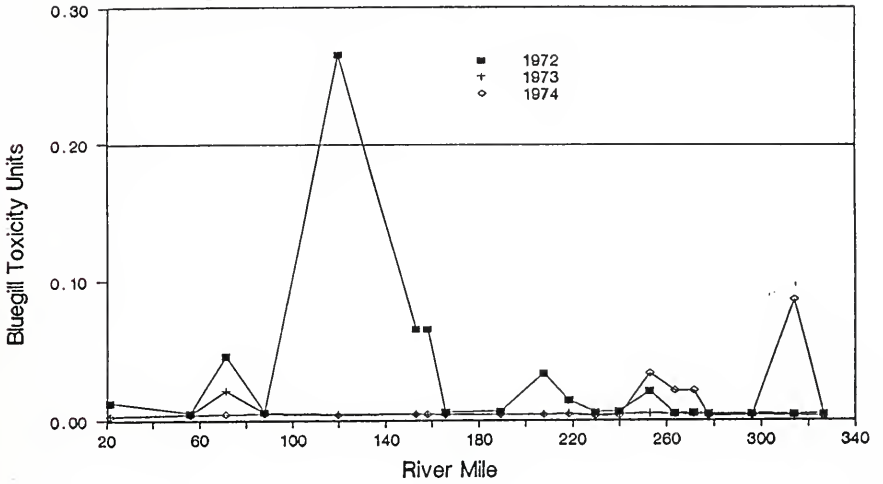
Illinois Waterway
Mean Toxicity Indices



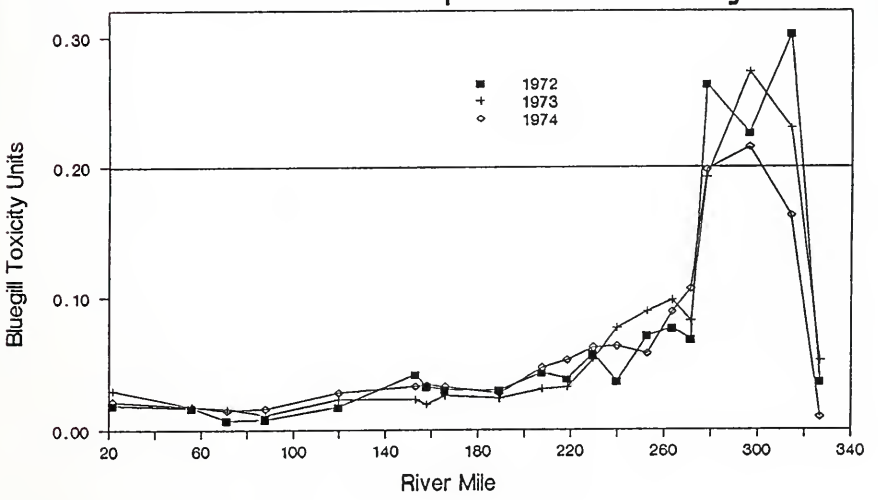
Illinois Waterway
Maximum Toxicity Indices



Illinois Waterway
Cyanide Component Toxicity

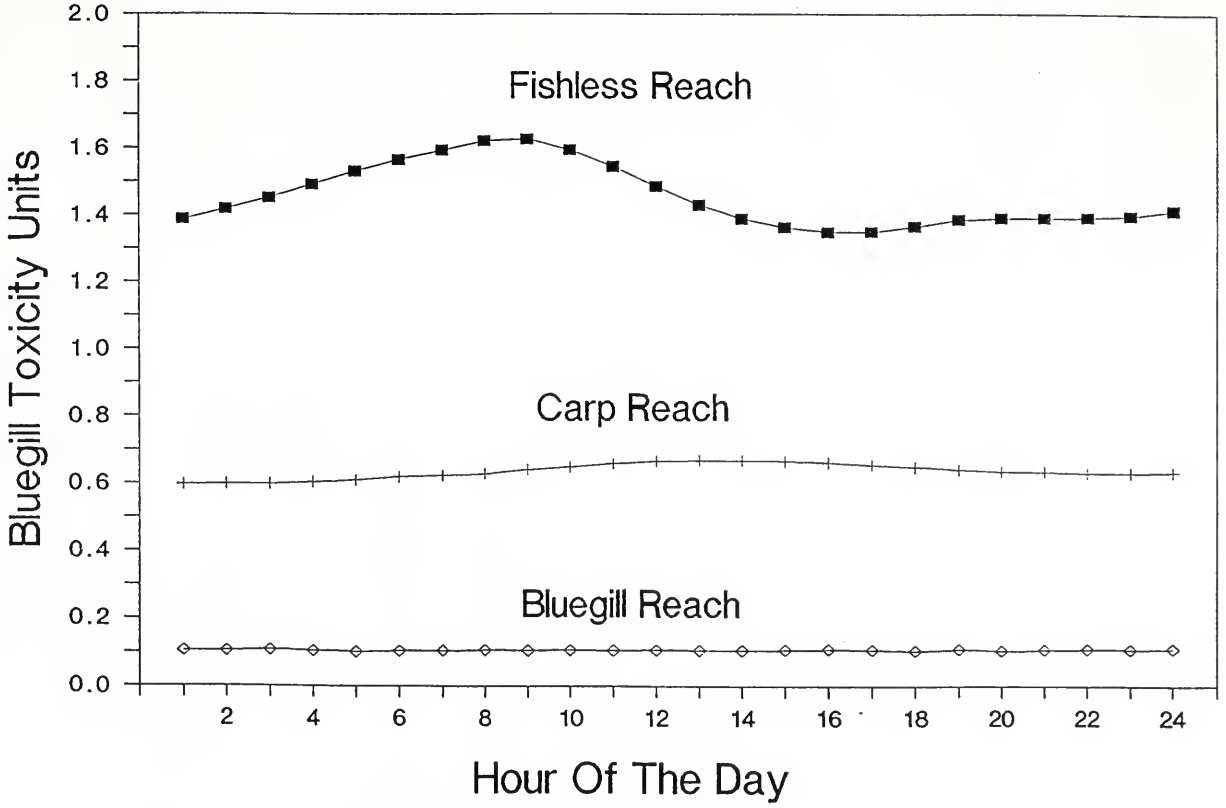


Illinois Waterway
Ammonia Component Toxicity



DuPage River Simulation

17 March 1972



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